Exhibit 3

Specific Causation Expert Report for Mark Cagiano
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PCE, vinyl chloride and Benzene. I have compared Mr. Cagiano's cumulative level of exposure to these toxins and note that his exposure was well above the amounts noted as toxic.

Viewing Dr. Reynolds' exposure numbers against these demonstrated toxic levels clearly establishes that Mr. Cagiano's exposure was significant and substantial. Mr. Cagiano exceeds each of the demonstrated levels identified by Dr. Bird. And he was stationed for well over the six quarters addressed in *Bove,* 2024.

VI. General Causation

Before advancing to the application of a differential etiology for Mr. Cagiano, it is important to first recognize whether there is enough evidence to establish whether the chemicals in the water at Camp Lejeune are capable of causing UTUC as a general matter. As addressed in my footnote regarding the studies above, because of a shared pathogenesis, I set out my overview of the bladder cancer science, which is a cancer of the urothelium, below and draw distinctions to and highlight the UTUC science specifically here and in my differential etiology below. Again, this is due to the common pathogenesis of these two cancers.

Numerous regulatory and scientific bodies have recognized that these four chemicals are toxic and capable of causing cancer. IARC recognizes TCE, VC, and benzene as having sufficient evidence for carcinogenicity in humans, and that that PCE is probably carcinogenic to humans.[73],[74] EPA concluded that "TCE is carcinogenic to humans by all routes of exposure," that is, by ingestion, inhalation, and dermal exposure.[75] Further, EPA PCE is "likely to be carcinogenic in humans by all routes of exposure" by EPA (2012). Similarly, the National Toxicology Program has recognized TCE as "a known human carcinogen" (2015) and PCE as "reasonably anticipated to be a human carcinogen." (2021a). ATSDR's 2017 Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases found sufficient evidence exists for PCE causing bladder cancer, stating that "the epidemiological studies provide sufficient evidence for causation and are consistent with the mechanistic information that certain genetic polymorphism may enhance the production of genotoxic PCE metabolites in the bladder via the GSH conjugate pathway."[76] While ATSDR 2017 did not find sufficient evidence for TCE and bladder cancer, later studies have strengthened the association as noted by Dr. Hatten. As

[73] International Agency for Research on Cancer. Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 2014;106:1-514.
[74] Environmental Protection Agency. Toxicological Review of Tetrachloroethylene (CAS No. 127-18-4). 2012
[75] EPA 2011
[76] Agency for Toxic Substances and Disease Registry. ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases. 2017:1-150.

reported by Dr. Hatten and Dr. Plunkett, epidemiological studies have identified elevated bladder cancer diagnoses associated with benzene and vinyl chloride.

As reported by Dr. Hatten, Dr. Plunkett, Dr. Gilbert, and Dr. Bird, both TCE and PCE share similar metabolic pathways: toxic metabolites are eventually excreted from the kidneys into urine where it sits in the bladder until voided. Dr. Plunkett identifies the same endpoint for benzene and vinyl chloride metabolites as well. This means that the toxic metabolites can spend hours in contact with urothelial cells inside the bladder and have contact with urothelial cells throughout the tract. Below is a figure from Dr. Gilbert explaining the metabolic pathways and outcome for TCE and PCE-induced bladder cancer, and I would extend this to include UTUC.

Dr. Gilbert reports that inhalation and dermal exposure from TCE-contaminated water at least doubles ingestion consumption figures (and with similar evidence for PCE). Dr. Gilbert further explains that a mixture of TCE, PCE, and benzene can produce additive effects that can cause bladder cancer in that both TCE and PCE share a similar metabolic pathway and all three chemicals promote chronic inflammation and immunosuppression. This is the shared pathogenesis.

Over time, the scientific consensus has progressed to greater certainty, and action, regarding the toxicity of the chemicals at Camp Lejeune. In December 2024 EPA finalized a rule banning on TCE and most commercial uses of PCE under the Toxic Substances Control Act, describing TCE as "extremely toxic" and PCE as "cancer-causing."[77] As noted by Dr. Bird in his supplemental report, "the EPA determined that any lesser restrictions on the use of TCE or PCE would fail to adequately protect public health." Dr. Bird further explained that EPA's safety measures were based on the wastewater concentrations, not consumption, meaning that the risk for those at Camp Lejeune (whose ingested concentrations alone are than the concentrations identified in the EPA rule) is even greater.

Accordingly, there is a sufficient basis to conclude that the chemicals in the water at Camp Lejeune are capable of causing bladder cancer. Again, I will point out more UTUC-specific support in my differential etiology below.

VII. Differential Etiology

UTUC arises from the cells lining the urinary system including the bladder, ureter, renal pelvis and prostatic urethra, most commonly in the transitional epithelium (also

[77] Environmental Protection Agency. Biden-Harris Administration Announces Latest Actions under Nation's Chemical Safety Law to Protect People from Cancer-Causing Chemicals Trichloroethylene and Perchloroethylene. December 9, 20214. https://www.epa.gov/newsreleases/ biden-harris-administration-announces-latest-actions-under-nations-chemical-safety-law.